British Heart Journal, 1977, **39**, 1299–1306

Echocardiography after reconstructive surgery for non-rheumatic mitral regurgitation[1]

RICHARD P. SNODGRASS, RICHARD E. KERBER, AND DAVID C. FUNK

From Cardiovascular Center and Cardiovascular Division, Department of Internal Medicine, University of Iowa, and Veterans Administration Hospitals, Iowa City, Iowa, U.S.A.

The echocardiographic appearances of the mitral valve after reconstructive surgery for non-rheumatic mitral regurgitation have been studied. Twenty-two patients, divided into 3 groups, were studied. In group 1 there were 12 patients with ruptured chordae tendineae and/or anterior mitral leaflet perforation, who underwent chordal reattachment and plication of leaflets or commissures. After operation common abnormalities were slow (<70 mm/s) diastolic EF slope (5 patients), multiple or dense echoes from valvular or subvalvular structures (5 patients), and abnormal posterior leaflet motion in diastole was observed in 3 patients. In group 2 there were 7 patients undergoing closure of an ostium primum atrial septal defect and primary repair by suture of a cleft anterior mitral leaflet. After operation slow EF slope occurred in 5 patients, but dense echoes were seen in only 1, and abnormal posterior leaflet echoes also in only 1. Group 3 consisted of 3 patients undergoing Carpentier ring insertion in the mitral annulus, all of whom showed dense echoes from the ring, with a slow EF slope in 2, and abnormal posterior leaflet motion in 2. Residual mitral regurgitation was common, as indicated by persistent mitral murmurs in 14. Systolic prolapse was not seen after operation. We conclude that echocardiographic abnormalities are common after reconstructive surgery for non-rheumatic mitral regurgitation. These abnormalities include multiple, dense valvular and subvalvular echoes, and abnormally slow and restricted diastolic motion of both leaflets suggesting that thickening, fibrosis, and decreased mobility of the valve apparatus occur because of the surgical plications and postoperative inflammatory changes. These structural changes were noted to be frequently accompanied by persistent mitral regurgitation.

The effect of operation on the mitral echogram has been evaluated previously, but such studies have concentrated on operations performed on rheumatic mitral valves (Mary *et al.*, 1973; Cope *et al.*, 1975), or on mitral replacement by various prostheses (Brodie *et al.*, 1976).

In mitral regurgitation of non-rheumatic origin, such as ruptured chordae tendineae caused by myxomatous degeneration or leaflet perforations secondary to bacterial endocarditis, mitral reconstructive surgery offers an alternative to valve replacement (Gerbode *et al.*, 1968; Selmonosky and Ehrenhaft, 1969; Manhas *et al.*, 1971). The ultrasonic appearances of the mitral valve after reconstructive procedures for non-rheumatic abnormalities have not been described in detail. The purpose of this study was to define the echocardiographic appearances after such mitral reconstructive surgery. We studied 3 groups of patients: patients with ruptured chordae tendineae and/or mitral leaflet perforation, patients with ostium primum atrial septal defect with a cleft anterior mitral leaflet, and patients undergoing Carpentier ring annuloplasty.

[1]Supported in part by NIH grants.
Received for publication 19 October 1976

Methods

All patients at the University of Iowa and Iowa City Veterans Administration Hospitals who had undergone mitral reconstructive surgery for non-rheumatic mitral regurgitation were identified by reviewing the surgical files. Some were being followed regularly in our clinics; others were contacted and invited to return for re-examination. For this study 22 patients (12 men and 10 women) who underwent operation between August 1965 and October 1975 were re-examined. Their ages at time of surgery ranged from 13 to 66 years. All patients stated that they were improved after the operation and all were considered to be in functional class I or

II (New York Heart Association) at the time of the examination.

All the patients were examined clinically and if a murmur was heard a phonocardiogram was recorded, using an Elema-Schonander 'mingograph' recorder. Echocardiograms were obtained using a Smith-Kline Ekoline ultrasonoscope and 2·25 mHz transducers, either unfocused or focused at 7·5 cm. Recordings were made on a Honeywell 1856 fibreoptic recorder. Recordings of the mitral valve were obtained using standard techniques (Feigenbaum, 1972). Standard measurements of left atrial dimension and left ventricular end-diastolic dimension (Feigenbaum, 1972) were made whenever the technical quality of the recordings permitted. The ventricular diameter was cubed to obtain left ventricular end-diastolic volume (Popp and Harrison, 1970; Pombo *et al.*, 1971). Since many of the patients had absent or paradoxical septal motion related to operation and/or volume overload, end-systolic volume or ejection fraction calculations would have been invalid. Measurements of the mitral valve diastolic closure velocity were made using the slope of the EF line. The normal EF slope in our laboratory is 70 to 150 mm/s. We also noted the presence or absence of multiple and/or dense valvular or subvalvular echoes, using normal sensitivity settings on the ultrasonoscope and attempting to relate density of the mitral echoes to those from the surrounding structures. The motion of the posterior leaflet of the mitral valve was considered abnormal if the leaflet failed to move posteriorly in early diastole (Duchak *et al.*, 1972).

Echocardiography was not routinely performed at the University of Iowa Hospital before 1971. Since many of our patients underwent reconstructive surgery earlier than this, preoperative echocardiograms were available for comparison in only 9 patients, and in 2 of these the records were from early studies using Polaroid photographs.

The patients were divided into 3 groups, depending on the type of operation. Group 1, consisting of 12 patients, had varying combinations of ruptured chordae tendineae to one or both mitral leaflets (9 patients), anterior leaflet perforation (4 patients), or papillary muscle dysfunction (1 patient). The aetiology of the chordal ruptures was considered to be myxomatous degeneration, while the perforations were the result of bacterial endocarditis. Operations on this group included varying combinations of reattachment of chordae (5 patients), plication of leaflets and/or commissures (10 patients), and pericardial patch or primary repair of perforations (3 patients). In group 2 the 7 patients all had ostium primum atrial septal defects with cleft anterior mitral leaflets. Operations in this group consisted of primary suture repair of the cleft leaflet plus pericardial or felt patch repair of the atrial septal defect. No chordal or other mitral apparatus operation was done in this group. Group 3 consisted of 3 patients: 1 with myxomatous degeneration of the mitral valve and 2 with papillary muscle dysfunction; all 3 underwent Carpentier ring annuloplasty. This is a procedure where a preformed, shaped ring is sewn into the mitral annulus in order to remodel the mitral valve, thereby reducing the annulus to its physiological dimensions and permitting better coaptation of the valve leaflets (Carpentier *et al.*, 1971). The mitral leaflets and suspensory apparatus were not directly operated upon in these 3 individuals.

Results

GROUP 1

The data from patients in group 1 are summarised in the Table. Of the 12 patients, 5 had preoperative echocardiograms. One showed systolic shuddering (Gramiak and Nanda, 1975) of the closed leaflet and 2 showed systolic prolapse (Fig. 1). One patient had a rapid EF slope (246 mm/s) and 1 had an EF slope below normal. Both patients in whom ventricular volumes could be calculated had large end-diastolic volumes (>160 ml).

After operation none of the patients had systolic prolapse or shuddering (Fig. 1). Abnormally strong or multiple echoes from valvular or subvalvular structures were present in 5 patients (Fig. 1 and 2). Posterior mitral leaflet motion in diastole was virtually absent in 3 patients (Fig. 1, 2, 3). The EF slope was rapid in 2 patients, normal in 5, and slow in 5. The mitral echogram appeared normal in 2 patients. The left ventricular volumes were large in 4 of the 6 patients in this group who had recordings adequate to calculate ventricular diameters. Four patients had an increased left atrial size ($>2·1$ cm/m^2). Of these 12 patients, 9 had persistent systolic murmurs (Fig. 4), including 1 of the 2 with a normal mitral echogram.

GROUP 2

The data from these patients also are summarised in the Table. Only 1 of these patients had a preoperative echocardiogram. After operation none had systolic prolapse, 5 of the 7 had slow EF slopes, and only 1 had abnormal posterior leaflet motion. Another had noticeably dense echoes from valvular and subvalvular structures (Fig. 5). Left ventricular volumes were enlarged in 2 of the 5 in whom this could be calculated. The left atrial size was increased in 3 patients. Five patients had persistent murmurs.

Table *Data of patients undergoing mitral valve reconstructive surgery*

Case No.	Sex	Age (y)	Diagnosis at operation	Preop. mitral valve echo: EF slope	Prolapse	LAD	EDV	Operation	EF slope	Prolapse	Postop. mitral valve echo: Dense valvular or subvalvular echoes	Posterior leaflet motion	LAD	EDV	Persistent MR murmurs
Group 1															
1	M	46	RCT-A and P	—	—	—	—	RA, P	58	None	Present	Normal	5·2	216	II/VI
2	F	30	ALP	—	—	—	—	CP, P	40	None	None	Abnormal	3·5	196	None
3	F	19	RCT-A and P	—	—	—	—	P	86	None	None	Normal	2·8	91	None
4	F	57	RCT-A, ALP	52	None	—	—	RA, P, CP	90	None	Present	Abnormal	—	—	III/VI
5	M	43	PMD	—	—	—	—	P	85	None	Present	Normal	3·7	220	IV/VI
6	M	37	RCT-A	150	None	—	—	RA, P	188	None	None	Normal	—	—	II/VI
7	M	45	RCT-P	—	—	—	—	RA	238	None	None	Normal	5·0	238	IV/VI
8	F	44	RCT-P	80	Present	—	—	P	72	None	Present	Abnormal	6·0	149	III/VI
9	M	54	RCT-P	246	Present	3·3	166	P	54	None	None	Normal	—	—	None
10	M	58	RCT-A	—	—	—	—	RA, P	40	None	None	Normal	—	—	II/VI
11	M	55	ALP, RCT-P	—	—	—	—	PP, P	74	None	None	Normal	4·0	—	I/VI
12	M	50	ALP	70	Shudder	3·9	250	PP, CP	30	No shudder	Present	Normal	3·0	—	I/VI
Group 2															
13	F	46	ASD, CAL	56	None	—	—	PD, S	62	None	None	Normal	3·1	—	II/VI
14	M	18	ASD, CAL	—	—	—	—	PD, S	80	None	None	Normal	2·6	125	II/VI
15	F	29	ASD, CAL	—	—	—	—	PD, S	62	None	None	Normal	2·7	—	None
16	F	63	ASD, CAL	—	—	—	—	PD, S	36	None	Present	Normal	5·7	227	V/VI
17	F	29	ASD, CAL	—	—	—	—	PD, S	58	None	None	Normal	5·0	110	II/VI
18	F	13	ASD, CAL	—	—	—	—	PD, S	80	None	None	Normal	2·2	64	None
19	M	16	ASD, CAL	—	—	—	—	PD, S	48	None	None	Abnormal	4·0	270	III/VI
Group 3															
20	F	66	MD	104	Present	5·3	118	CR	50	None	Present	Abnormal	5·3	196	None
21	M	62	PMD	128	None	5·3	314	CR	80	None	Present	Abnormal	4·8	275	None
22	M	64	PMD	125	None	—	—	CR	60	None	Present	Normal	4·0	—	None

Abbreviations

RCT, ruptured chordae tendineae supplying anterior (A) or posterior (P) leaflets; PMD, papillary muscle dysfunction; ALP, anterior leaflet perforation; ASD, atrial septal defect (ostium primum type); CAL, cleft anterior leaflet; RA, reattachment of chordae; P, plication of leaflets and/or commissures(s); CP, primary suture repair of perforation; PP, pericardial patch repair of perforation; PD, pericardial or felt patch repair of ASD; S, suture repair of leaflet cleft; MD, myxomatous degeneration of mitral valve; LAD, left atrial dimension; EDV, left ventricular end-diastolic volume; MR, mitral regurgitation; CR, Carpentier ring annuloplasty.

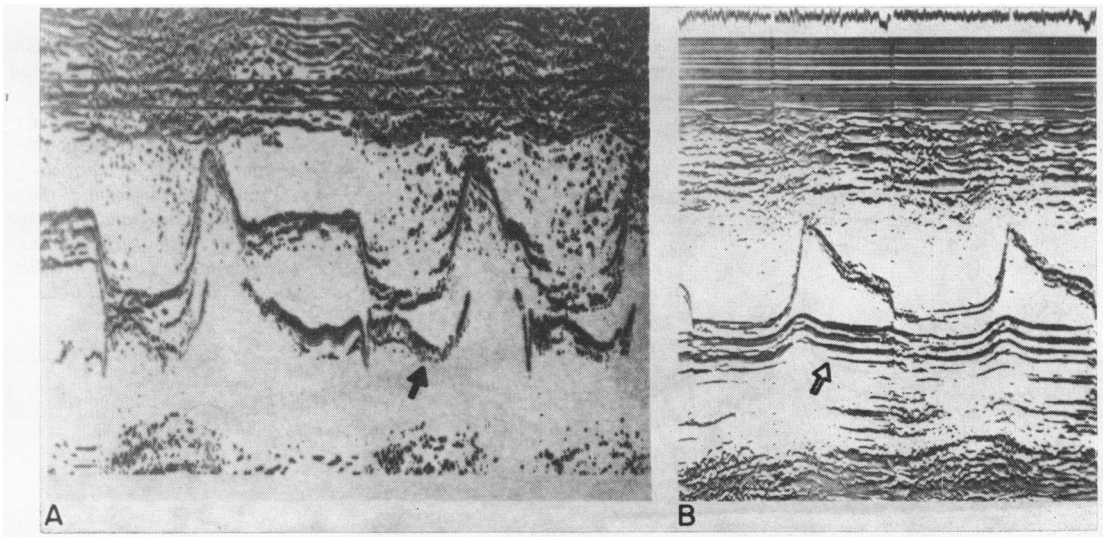

Fig. 1 *Echocardiograms from a patient with ruptured chordae tendineae and redundant posterior leaflet. (A) Preoperative echocardiogram, showing midsystolic mitral leaflet prolapse (closed arrow). (B) Postoperative echocardiogram, after posterior leaflet plication. Dense, multiple echoes from the posterior mitral leaflet (open arrow) are present, and the leaflet moves minimally in diastole. No prolapse is present.*

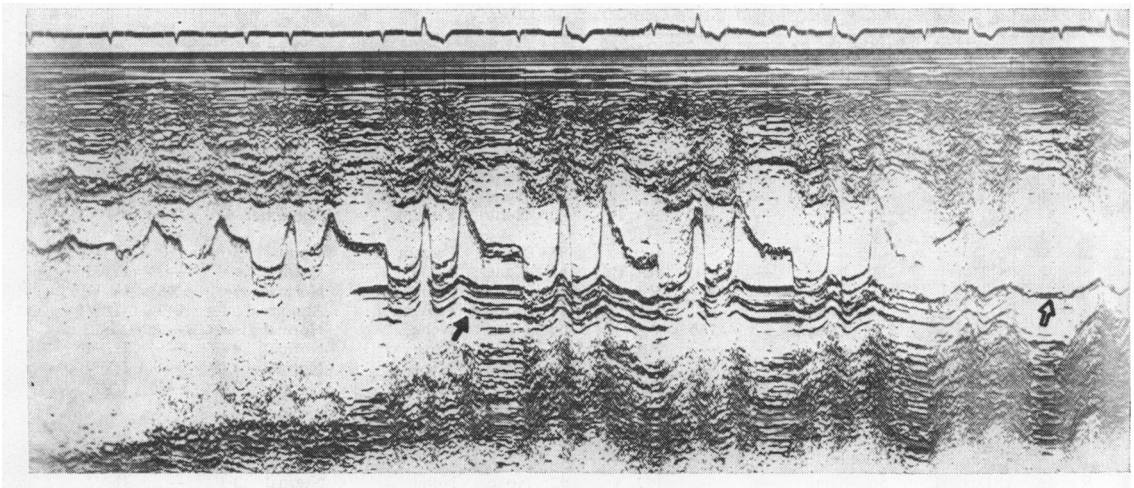

Fig. 2 *Postoperative echocardiographic sweep from the same patient as in Fig. 1. Dense, multiple echoes from the immobile posterior leaflet (closed arrow) are present. The echoes from the chordae tendineae (open arrow) are normal.*

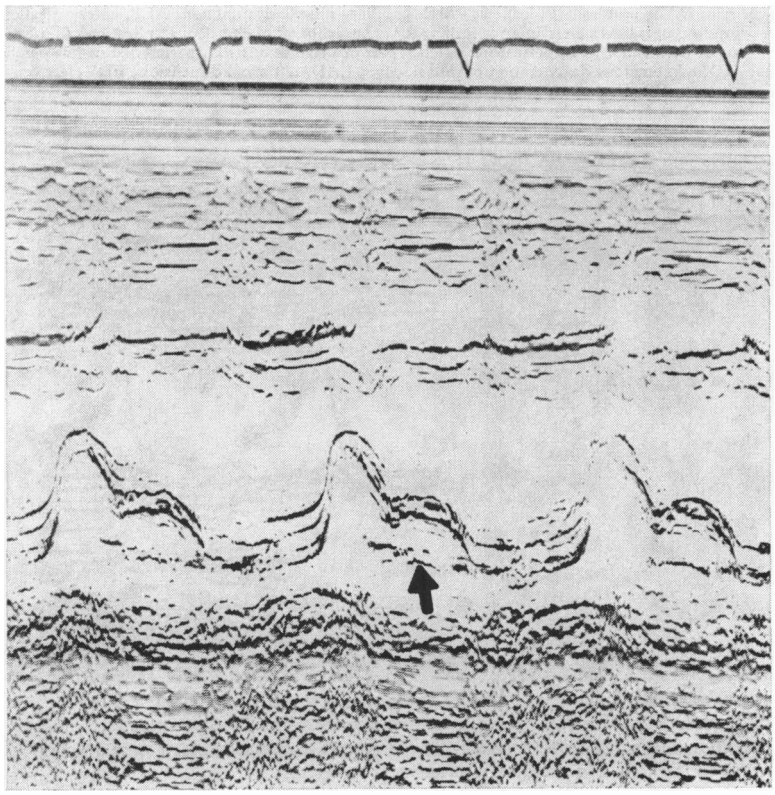

Fig. 3 *Postoperative echocardiogram from a patient who had had subacute bacterial endocarditis, ruptured chordae tendineae, and two anterior leaflet perforations. Operation consisted of reattachment of chordae, plication of the anterior commissure, and primary suture repair of the perforations. The motion of the posterior leaflet (arrow) is abnormal in diastole.*

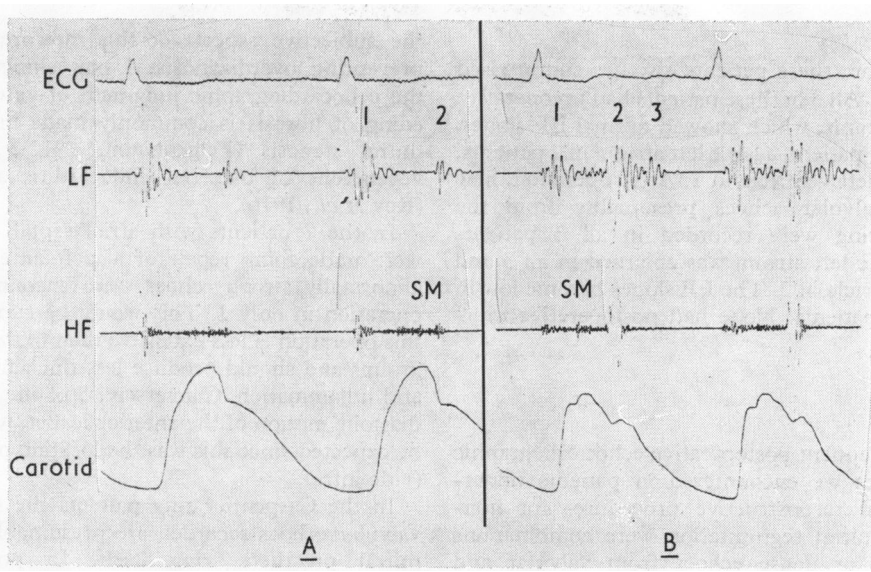

Fig. 4 *(A) Postoperative phonocardiogram from the patient shown in Fig. 1. A mid to late systolic murmur is present. (B) Postoperative phonocardiogram from the patient shown in Fig. 3. A holosystolic murmur and a third heart sound are present. Both recordings were made at the apical area. LF, low frequency band-pass filter; HF, high frequency filter; SM, systolic murmur.*

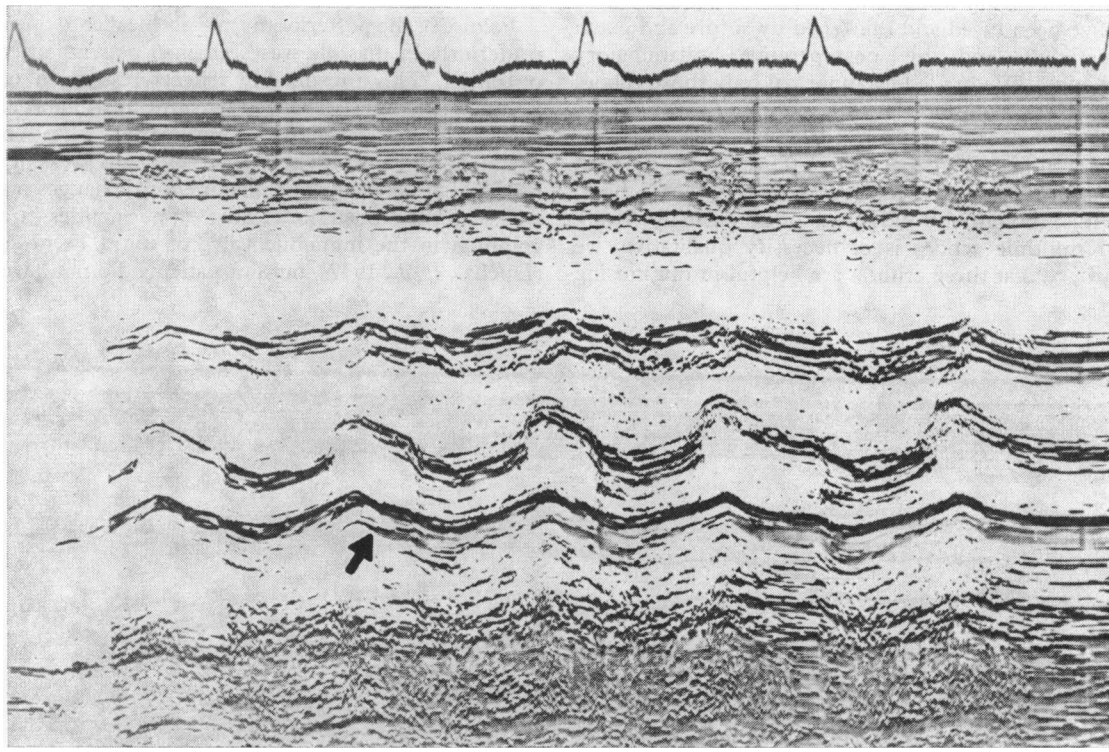

Fig. 5 *Postoperative echocardiogram from a patient with an ostium primum atrial septal defect and a cleft anterior mitral leaflet. Operation involved closure of the atrial septal defect and primary repair by suture of the cleft leaflet. The EF slope is slow, and there are dense, multiple echoes from the anterior mitral leaflet and the chordae tendineae (arrow).*

GROUP 3

The data from these patients also are summarised in the Table. All 3 of these patients had preoperative echocardiograms, which showed normal EF slopes, prolapse in 1 patient, a large left atrium in 2 patients, and a large left ventricle in 1. After operation, new dense subvalvular echoes presumably from the prosthetic ring were recorded in all 3 patients (Fig. 6). The left atrium was enlarged in all 3 and the left ventricle in 2. The EF slopes became low in 2 of the 3 patients. None had postoperative murmurs.

Discussion

The most frequent postoperative echocardiographic abnormalities we encountered in patients undergoing mitral reconstructive procedures for nonrheumatic mitral regurgitation were combinations of multiple or dense echoes from valvular and subvalvular structures, abnormally restricted diastolic posterior leaflet motion, and reduced EF slope of the anterior leaflet. The strong, dense echoes are presumably the result of increased ultrasonic reflectance from leaflet and chordal structures which have been fused and thickened by suture and patch materials and by postoperative inflammatory changes. We graded as abnormal only those echoes that were noticeably stronger than adjacent echoes when the sensitivity settings of the ultrasonoscope were adjusted so that the left ventricle did not display an excessive number of intracavitary echoes. Thus, though the judgment of abnormally strong or multiple echoes is of necessity qualitative, we believe that these criteria are helpful in minimising the subjective aspects of this interpretation, and preventing overdiagnosis. Using similar methods the echocardiographic judgment of valvular thickening of fibrosis is commonly made in rheumatic mitral stenosis (Feigenbaum, 1972), and when vegetations of bacterial endocarditis are present (Roy *et al.*, 1976).

In the 7 patients with atrial septal defect who were undergoing repair of a cleft anterior leaflet, abnormally strong echoes were encountered after operation in only 1. This would be expected since this operation is less extensive than in the other two groups and should produce less thickening, fusion, and inflammation. Conversely, 5 of the 7 had slow diastolic motion of the anterior leaflet, which might be expected since this was the location of the repair (vide infra).

In the Carpentier ring patients the strong subvalvular echoes recorded are presumably from the mitral prosthetic ring itself. In position and appearance these echoes were similar to the dense echoes which have been noted in patients with mitral annular calcification (Hirschfeld and Emilson, 1975), where the calcium occupies an anatomical position analogous to the Carpentier ring.

Failure of the posterior mitral valve leaflet to open posteriorly in diastole was seen in 6 patients after operation. This presumably reflects restriction of normal leaflet motion caused by suturing of chordal and cuspal tissue and commissural plication (Selmonosky and Ehrenhaft, 1969) and possibly because of subsequent inflammatory changes as well. Similar posterior leaflet abnormalities are common in the immobile valve of mitral stenosis (Duchak *et al.*, 1972), but our patients did not have

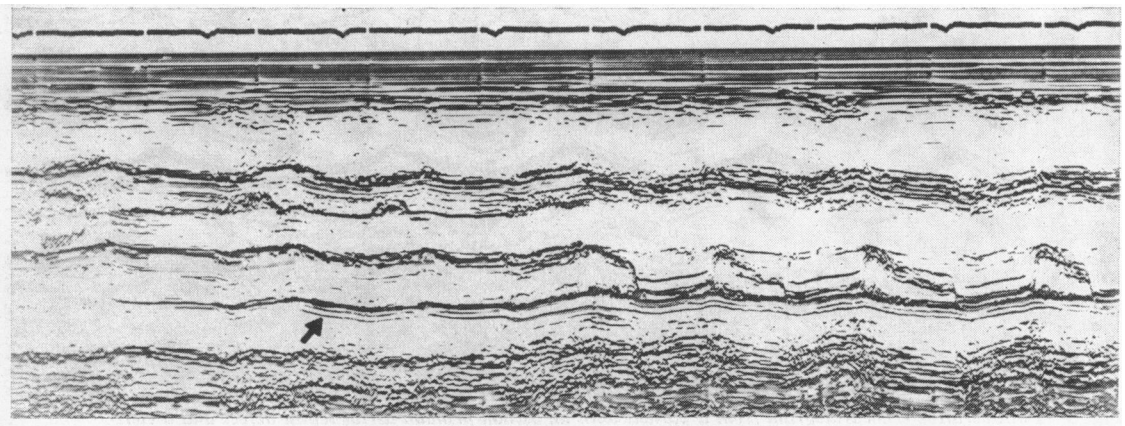

Fig. 6 *Postoperative echocardiographic sweep from a patient with papillary muscle dysfunction (coronary artery disease), after insertion of a Carpentier ring in the mitral annulus. Dense multiple echoes from the ring (arrow) are present and the posterior leaflet motion is abnormal in diastole.*

rheumatic disease and none had such abnormal posterior leaflet motion preoperatively.

A decreased EF slope—early diastolic closure velocity—was encountered postoperatively in 13 of these 22 patients, whereas this was seen in only 2 of the 9 available preoperative echoes. Several explanations for this are possible. Zaky *et al.* (1968) showed that the initial rate of descent of the anterior mitral leaflet (EFo slope) is primarily related to the early diastolic movement of the mitral ring; if there is a decreased rate of ventricular filling the mitral annulus will recede more slowly from the ultrasound transducer and hence the EF slope of the anterior leaflet mitral valve, determined by annular motion, would be reduced. Patients with reduced left ventricular compliance and hence slow ventricular filling would, therefore, be expected to have a decreased EF slope, and this is commonly seen in conditions such as aortic stenosis or hypertrophic obstructive cardiomyopathy (Duchak *et al.*, 1972; Popp and Harrison, 1969). Quinones *et al.* (1974), confirmed that a decreased mitral valve slope was associated with abnormalities of left ventricular diastolic pressure-volume relation. A decrease in the amount of mitral regurgitation because of operation should indeed reduce the rate of left ventricular filling, but only to the normal range. There is no *a priori* reason to suppose that operation would cause a chronic decreased rate of ventricular filling and low EF slope. In fact, 6 of the 12 patients with a low EF slope had residual mitral regurgitation, which would be expected to increase diastolic filling and result, if anything, in a rapid EF velocity. Thus, abnormally slow ventricular filling does not seem a likely explanation for the commonly encountered low EF slope in those patients.

Patients with mitral stenosis typically have slow EF slopes; this is the result of a combination of a thickened and immobile valve and slow ventricular filling caused by mitral obstruction (Zaky *et al.*, 1968). In the absence of rheumatic heart disease significant mitral orifice obstruction is not commonly produced by the type of reconstructive operation which our patients underwent (Selzer *et al.*, 1972), and none had diastolic rumbles audible or recordable after operation. However, postoperative cardiac catheterisation data are not available and we cannot entirely exclude the possibility that at least some degree of postoperative mitral obstruction was present in some patients. A more likely explanation, however, is that the low postoperative EF slope was the result of limitation of motion of the valve leaflet itself, resulting from the suturing and plication procedures which were done. The steeper part of the mitral diastolic descent (Fo-F slope) was the result of leaflet motion independent of annular movement (Zaky *et al.*, 1968); if this portion of the diastolic motion were reduced owing to partial leaflet immobilisation the net measured EF slope would be decreased.

Persistent regurgitant murmurs were present in 14 of our patients; previous investigators have also found a high incidence of postoperative murmurs in patients similar to ours (Manhas *et al.*, 1971; Selzer *et al.*, 1972). Systolic leaflet prolapse was not detected echocardiographically in any of our postoperative patients and thus prolapse cannot be a common cause of mitral regurgitation after reconstructive operations. Rather, the echocardiographic findings suggest that the postoperative regurgitation in these patients is caused by failure of the thickened and relatively immobile valve leaflets to coapt properly. In only 2 of the patients with murmurs was the mitral echogram entirely normal.

Left ventricular size was increased in 8 patients, 5 of whom had regurgitant murmurs. Left atrial size was increased in 9 patients, of whom 7 had murmurs. It seems likely that these high volumes were caused by significant mitral regurgitation.

We conclude that reconstructive operations for non-rheumatic mitral regurgitation frequently result in echocardiographic appearances suggestive of thickening, fibrosis, and decreased mobility of the valve apparatus, probably the result of a combination of surgical plications and postoperative inflammatory changes. These structural changes, rather than leaflet prolapse, are the likely cause of the persistent mitral regurgitation and left ventricular and atrial enlargement encountered in many patients undergoing this type of mitral operation. In addition to identifying the nature of the persistent valvular dysfunction in these individuals, this study should alert the echocardiographer to the possible confusion of these findings with the similar appearance of the rheumatic mitral valve. Exact information on the nature of previous mitral operations assists in the interpretation of postoperative echocardiograms.

References

Brodie, B. R., Grossman, W., McLaurin, L., Starek, P. J. K., and Craige, E. (1976). Diagnosis of prosthetic mitral valve malfunction with combined echo-phonocardiography. *Circulation*, **53**, 93–100.

Carpentier, A., Deloche, A., Dauptain, J., Soyer, R., Blondeau, P., Piwnica, A., and Dubost, C. (1971). A new reconstructive operation for correction of mitral and tricuspid insufficiency. *Journal of Thoracic and Cardiovascular Surgery*, **61**, 1–13.

Cope, G. D., Kisslo, J. A., Johnson, M. L., and Behar, V. S. (1975). A reassessment of the echocardiogram in mitral stenosis. *Circulation*, **52**, 664–670.

Duchak, J. M., Chang, S., and Feigenbaum, H. (1972). The posterior mitral valve echo and the echocardiographic diagnosis of mitral stenosis. *American Journal of Cardiology*, **29**, 628–632.

Feigenbaum, H. (1972). *Echocardiography*. Lea and Febiger, Philadelphia.

Gerbode, F., Hill, J. D., Kelly, J. J., Jr., Selzer, A., and Kerth, W. J. (1968). Surgical correction of mitral insufficiency due to ruptured chordae tendineae. *Circulation*, **37-38**, Suppl II, II-119–123.

Gramiak, R., Nanda, N. C. (1975). Mitral valve. In *Cardiac Ultrasound*, pp. 47–73. Ed. by R. Gramiak and R. C. Waag. Mosby, St. Louis.

Hirschfeld, D. S., and Emilson, B. B. (1975). Echocardiogram in calcified mitral anulus. *American Journal of Cardiology*, **36**, 354–356.

Manhas, D. R., Rittenhouse, E. A., Hessel, E. A., and Merendino, K. A. (1971). Reconstructive surgery for the treatment of mitral incompetence. *Journal of Thoracic and Cardiovascular Surgery*, **62**, 781–787.

Mary, D. A. S., Pakrashi, B. C., Wooler, G., and Ionescu, M. I. (1973). Study with reflected ultrasound of patients with mitral valve repair. *British Heart Journal*, **35**, 480–487.

Pombo, J. F., Troy, B. L., and Russell, R. O. (1971). Left ventricular volumes and ejection fraction by echocardiography. *Circulation*, **43**, 480–490.

Popp, R. L., and Harrison, D. C. (1969). Ultrasound in the diagnosis and evaluation of therapy of idiopathic hypertrophic subaortic stenosis. *Circulation*, **40**, 905–914.

Popp, R. L., and Harrison, D. C. (1970). Ultrasonic cardiac echography for determining stroke volume and valvular regurgitation. *Circulation*, **41**, 493–502.

Quinones, M. A., Gaasch, W. H., Waisser, E., and Alexander, J. K. (1974). Reduction in the rate of diastolic descent of the mitral valve echogram in patients with altered left ventricular diastolic pressure-volume relations. *Circulation*, **49**, 246–254.

Roy, P., Tajik, A. J., Giuliani, E. R., Schattenberg, T. T., Gau, G. T., and Frye, R. L. (1976). Spectrum of echocardiographic findings in bacterial endocarditis. *Circulation*, **53**, 474–482.

Selmonosky, C. A., and Ehrenhaft, J. L. (1969). The technique of open mitral reconstruction in the treatment of acquired and congenital mitral incompetence. *Journal of Cardiovascular Surgery*, **10**, 426–430.

Selzer, A., Kelly, J. J., Jr., Kerth, W. J., and Gerbode, F. (1972). Immediate and long-range results of valvuloplasty for mitral regurgitation due to ruptured chordae tendineae. *Circulation*, **45-46**, Suppl. I, I-52–56.

Zaky, A., Nasser, W. K., and Feigenbaum, H. (1968). A study of mitral valve action recorded by reflected ultrasound and its application in the diagnosis of mitral stenosis. *Circulation*, **37**, 789–799.

Requests for reprints to Dr. Richard E. Kerber, Cardiovascular Division, University of Iowa Hospitals, Iowa City, Iowa 52242, U.S.A.